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ORTHOSTATIC RESPONSES TO DIETARY SODIUM RESTRICTION DURING HEAT ACCLIMATION

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ABSTRACT

Several studies have shown that individuals consuming low-salt diets and working in the heat have an increased risk or incidence of heat injury, suggestive of inadequate cardiovascular adjustment. Furthermore, others have shown that prolonged work in hot climates can precipitate orthostatic hypotension and syncope. This study was designed to evaluate the effects of moderate-salt (MS) and low-salt (LS) diets on the circulatory responses and incidence of presyncopal symptoms to an orthostatic test (OT) during successive days of heat acclimation (HA). Seventeen unacclimatized male soldiers (mean ± SE: age, 20±1 yrs) participated in this two-phase study. The first phase consisted of a seven day dietary stabilization period during which all subjects consumed similar diets of about 4000 kcal/day containing 8g NaCl and lived in a dormitory setting (21°C, 30% RH). The second phase commenced on day eight and consisted of dietary NaCl restriction and 10 days HA (days 8-17). Volunteers were randomly assigned to either the MS diet (n=9) providing 8g NaCl/day or the LS diet (n=8) furnishing just 4g NaCl/day. The acquisition of HA was manifested in both groups by reductions in exercising rectal temperature and heart rate (HR); these characteristics were similar in the MS and LS diets. The OT was performed at 21°C on day seven of the stabilization phase and on days 9, 11, 13, 15, and 17 of the HA phase, before and after 8.5 hr of intermittent treadmill walking (30 min/hr, 5.6 km/hr, 5% grade) in a hot environment (41°C, 21% RH, 1.2 m/sec windspeed). Blood pressure (BP) and HR responses at 1, 2 and 4 min and any presyncopal symptoms were recorded after assuming an upright position from recumbency. All subjects completed the OT before and after prolonged exercise in the heat without incidence of either hypotension or presyncopal symptoms irrespective of dietary-salt intake and day of HA. There were no between-diet or between-day differences in supine systolic (Psys, 117±1 mmHg), diastolic (52±1 mmHg), and mean (74±1 mmHg) BP or HR (66±1 bpm) during the pre-exercise OT. Furthermore, the pre-exercise BP and HR responses to standing were not different between MS and LS diets or between HA days, and these OTT responses reflected typical increments in HR (17±1 bpm), Psys (4±1 mmHg), Pdias (18±1 mmHg), and mean BP (13±1 mmHg). The post-exercise OT responses were qualitatively similar to those observed pre-exercise. However, during the post-exercise OT on day 11, significant between-diet differences occurred; smaller increases were observed in the LS group in Pdias (LS: 4±2 mmHg; MS: 22±2 mmHg) and mean BP (LS: 6±4 mmHg; MS: 18±1 mmHg) at 2 and 4 min standing. The plasma volume expansion (%ΔPV) associated with HA was also significantly attenuated in the LS diet (+2%, day 11; +7%, day 15) compared to the MS diet (+11.5%, day 11; +13%, day 15), although all sweat losses incurred during HA were replaced hourly with equal volumes of pure water. These results indicate that the prolonged work in the heat can be performed without orthostatic hypotension or syncope while consuming 4g NaCl/day with adequate fluid replacement. Furthermore, the circulatory responses to OT showed no improvement with successive days of HA irrespective of dietary-salt intake.

INTRODUCTION

During rapid deployment from garrison to either jungle or desert conditions, both caloric and salt intakes of soldiers are typically reduced. Concomitant with these reductions in consumption are the elevated sweat NaCl losses, particularly during the first few days of heat exposure. Although the salt requirements for extended living and working in a hot environment have been previously studied, the results are of limited value because requirements range from 2g per day to as high as 20g per day (1,2,3,9,13,14,15,16). As reported by Hubbard et al. (9), the lack of agreement between studies most probably is due to inadequate fluid replacement (dehydration), uncontrolled exercise level, unknown heat acclimation status, unknown initial salt and water status, and lack of a dietary stabilization period.

Orthostatic hypotension and/or presyncopal symptoms have been reported following numerous stressors including water deprivation, heat exposure and dehydration due to exercise (4,7,8). In addition, both prolonged work in hot climates or acute exhaustive exercise can precipitate orthostatic hypotension and syncope (5). Furthermore, several studies have shown that individuals consuming low-salt diets and working under hot conditions may have an increased risk of heat injury (1,3) or incidence of orthostatic hypotension (14,15) and syncope (3,14,15), suggestive of inadequate circulatory adjustment.

It is generally agreed that heat acclimation is manifested by improved cardiovascular and thermoregulatory responses to work in a hot environment. In contrast, the effects of heat acclimation on orthostatic responses are inconsistent with both positive (11,12) and negative benefits (4,7) reported. Mild orthostatic increases in heart rate and decreases in blood pressure are typical clinical signs of acute moderate NaCl losses (16). However, the effects of a reduction in salt intake on orthostatic responses during repeated days of working in the heat are largely unknown, but are important to the military planner particularly during the first few days of heat exposure when both the incidence of syncope and the salt loss in sweat are high.

The current study, which incorporated seven days of dietary stabilization followed by 10 days of heat acclimation while consuming a low-salt (4g NaCl/day) diet, afforded the unique opportunity to assess the impact of reduced NaCl intake on orthostasis during subsequent days of heat exposure. In addition, the effect of heat acclimation on orthostatic responses was evaluated.

METHODS

Seventeen (17) healthy, physically fit, unacclimatized male soldiers (mean $\pm$SE: age, 20$\pm$1 yrs; height, 179$\pm$2 cm; weight, 78.4$\pm$2.4 kg; surface area, 1.97$\pm$0.03 m^2) were briefed on the experimental design, procedures, medical risks, and freedom to withdraw at any time without retribution. Prior to participation, a medical history and physical examination were performed, and the volunteers provided their written consent.

This study consisted of two phases. The first phase comprised a seven day dietary stabilization period during which all subjects consumed diets of about 4000 kcal/day containing 8g NaCl and lived in a dormitory setting (21°C, 30% relative humidity). The second phase commenced on day eight and consisted of dietary NaCl restriction and 10 days of heat acclimation (days 8-17). Subjects were randomly assigned to either the moderate-salt (MS) diet (n=9) and continued to consume 8g NaCl/day or to a low-salt (LS) diet (n=8) and consumed 4g NaCl/day. During the 17 days, dietary intake and activity level were supervised 24 hrs/day.

Heat acclimation to a simulated desert climate was acquired via exercise in the heat for 10 consecutive days (days 8-17). On each day, subjects performed eight repetitions of intermittent treadmill walking (30 min/hr, 5.6 km/hr, 5% grade) and rest (30 min/hr) in a hot environment (41°C, 21% RH, 1.2 m/sec wind). Subjects unable to complete all work/rest cycles remained in the hot environment. After

completion of the 8.5 hr exercise-heat exposure, subjects returned to their dormitory setting (21°C) until resuming the heat exposure the next morning.

During the heat acclimation trials, ad libitum fluid intake and body weight were measured every 30 min. Sweat loss (SL) was calculated as the difference between the pre- and post-exercise nude body weights, adjusted for fluid and food intake, urination, blood sampling, and respiratory water loss. Changes in plasma volume (PV) were calculated using venous hematocrit and hemoglobin samples drawn before the first treadmill iteration and immediately upon completing the final walk.

An orthostatic test (OT) was performed in a secluded room at 21°C on day seven of the stabilization phase, and on days 9, 11, 13, 15, and 17 of the heat acclimation phase, both before and after the 8.5 hr exercise-heat exposure. The purpose of the OT was to measure the circulatory responses to a sudden change in position from horizontal to upright. Specifically, each subject lay supine for 4-10 min, and then quickly stood from the lying to erect (90°) position by his own effort. Blood pressure (systolic, diastolic and mean) and heart rate were measured just prior to standing and at 1, 2 and 4 min after assuming an upright position. While erect, body weight was supported by the legs. Appearance of presyncopal symptoms (lightheadedness, dizziness, nausea, vomiting, or pallor) constituted orthostatic intolerance.

Data were analyzed using analysis of variance with repeated measures (ANOVA) with Scheffe's post hoc analysis. The null hypotheses were rejected at $p<0.05$. Data are expressed as mean $\pm$ standard error (1SE).

RESULTS

The acquisition of heat acclimation (HA) was manifested in both groups by reductions in exercising heart rate (HR, 154$\pm$6 bpm to 125$\pm$3 bpm, $p<0.005$) and rectal temperature (Tre, 38.3$\pm$0.1°C to 37.8$\pm$0.1°C, $p<0.02$) from day 8 to day 17. These changes were similar in the moderate-salt (MS) and low-salt (LS) diets. Exercising mean blood pressures (BP, 73$\pm$1 mmHg) and daily sweat losses (5.2$\pm$0.1L) incurred by HA were not different between diets or days. These daily fluid losses were replaced hourly during the exercise-heat regimen by having each subject consume a volume of pure water that matched the deficit in body weight; thus, negligible body weight losses (0.03$\pm$0.006 kg/hr) were observed. Nonetheless, the plasma volume expansion (%ΔPV) typically associated with HA was significantly ($p<0.04$) attenuated on days 11 and 15 in the LS diet compared to the MS diet, although the %ΔPV incurred during the eight hours of exercise was similar (+6.5$\pm$0.6%) for both diets on all days (TABLE 1). No heat injuries occurred in either diet group although the daily eight hrs of walking resulted in several cases of overuse injury (blisters, shin splints, skin chafing) and completion of only 66$\pm$4 and 60$\pm$7 out of a possible 80 walks for the MS and LS diets, respectively.

Neither diet nor day of HA had an effect on the values for supine HR and systolic (Psys), diastolic (Pdias), or mean blood (BP) pressures during the pre-exercise OT. All subjects completed the pre-exercise OT without any presyncopal symptoms or orthostatic hypotension. In addition, the pre-exercise OT responses from supine to 1, 2 and 4 min standing were not different between the MS and LS diets or between the 10 days of HA (TABLE 2), and reflected typical average increments in HR (17$\pm$1 bpm), Psys (4$\pm$1 mmHg), Pdias (18$\pm$1 mmHg), and mean BP (13$\pm$1 mmHg).

Irrespective of dietary-salt intake or day of HA, all subjects completed the post exercise-heat exposure OT without displaying signs or symptoms of intolerance. No significant differences in the post-exercise supine values for HR, Psys, Pdias and BP were seen between either diets or days of HA. While

TABLE 1. Responses to Heat Acclimation

		Day 8	Day 11	Day 15	Day 17
Final walk	4g	147±8	130±7	126±6 (4)	131±6
HR (bpm)	8g	161±8	135±4‡	126±4‡ (8)	128±3‡
Pre-exercise	4g	37.3±0.1	37.1±0.1	36.9±0.2‡ (4)	37.0±0.1
Tre (°C)	8g	37.4±0.1	37.1±0.1	37.0±0.1‡ (8)	37.2±0.1*
Final walk	4g	38.3±0.2	38.1±0.1	37.7±0.1‡ (4)	37.8±0.1‡
Tre (°C)	8g	38.3±0.1	38.0±0.1	37.8±0.1‡ (8)	37.9±0.1
Daily Sweat	4g	6.36±0.25	5.45±0.39	4.49±0.36‡	5.53±0.32
Loss (L)	8g	5.26±0.48*	5.87±0.25	5.24±0.41	5.61±0.24
Final walk	4g	77±3	74±4 (6)	70±6 (4)	69±4
BP (mmHg)	8g	77±3 (8)	72±4 (5)	75±2 (8)	68±2
% ΔPV[1]	4g	-----	2.0±1.8	6.6±2.0	12.5±1.6†
	8g	-----	11.5±2.8*	12.8±2.2*	12.4±1.7
Daily % ΔPV[2]	4g	8.0±1.3	4.8±1.2	7.8±4.5	5.7±1.7
	8g	8.4±2.2	6.2±1.8	4.8±1.1	6.2±0.6

HR, heart rate; Tre, rectal temperature; BP, blood pressure
Values are mean ± SE. Unless indicated, LS (4g) diet, n=8; MS (8g) diet, n=9.
1 Percent change in plasma volume from day 8 pre-exercise to pre-exercise that day.
2 Percent change in plasma volume from pre-exercise that day.
* Significantly different ($p<0.05$) from LS (4g) diet; † from day 11; ‡ from day 8.

TABLE 2. The Effects of Exercise and Heat-Acclimation on Orthostatic Responses

			Change upon standing (4min) from the supine position				
		Supine	Day 9	Day 11	Day 13	Day 15	Day 17
Heart Rate (bpm)							
Pre-exercise	4g	66±2	17±2	17±4	24±3	16±2	19±2
	8g	66±1	19±7	14±7	15±7	11±5	18±4
Post-exercise	4g	70±1	19±4	20±3	16±2	16±3	11±3
	8g	71±2	16±3	13±2	18±4	14±6	14±5
Psys (mmHg)							
Pre-exercise	4g	113±1	3±4	-3±5	1±4	11±4	3±3
	8g	122±1	7±5	8±5	6±3	4±2	6±3
Post-exercise	4g	111±1	1±1	8±8	0±6	7±6	-1±3
	8g	113±1	9±6	11±3	5±3	6±3	2±3
Pdias (mmHg)							
Pre-exercise	4g	52±1	21±7	18±3	13±3	21±3	20±3
	8g	51±1	18±4	18±6	18±3	18±3	12±5
Post-exercise	4g	58±1	17±6	4±5	18±4	13±5	20±4
	8g	57±1	17±4	22±2*	17±2	24±5	10±4
BP (mmHg)							
Pre-exercise	4g	72±1	15±6	11±2	9±3	18±3	14±3
	8g	75±1	15±3	15±6	14±3	13±3	10±3
Post-exercise	4g	76±1	12±4	6±5	12±3	11±4	13±3
	8g	76±1	14±2	19±1*	13±1	18±3	8±3

* Significantly different ($p<0.05$) from LS (4g) diet.

increases in HR and blood pressures at 1, 2 and 4 min standing were generally not different between diets or HA days, the OT response was significantly attenuated in the LS diet on day 11 (day four of exercise-heat exposure). During the post-exercise OT on day 11, subjects on the LS diet manifested significantly smaller increases in Pdias (LS: 4±2 mmHg; MS: 22±2 mmHg, $p<0.02$) and mean BP (LS: 6±4 mmHg; MS: 18±1 mmHg, $p<0.007$) concurrent with larger increments in HR (LS:20±3 bpm; MS:13±2 bpm) at 2 and 4 min standing.

The OT responses following the 8.5 hr exercise-heat exposure were qualitatively similar to those seen pre-exercise. Significantly ($p<0.03$) higher post-exercise supine values for Pdias and mean BP were seen in the LS diet on days 11 and 15 and for HR, Psys and Pdias in the MS diet on day 17. A further important finding was the smaller ($p<0.02$) increase in both Pdias and mean BP in the LS diet following the exercise-heat exposure on day 11 compared to the pre-exercise OT response.

DISCUSSION

The work of Taylor et al. (14,15) has shaped our opinions and much doctrine regarding salt requirements for preventing performance decrements and heat injury in hot climates. This classical study demonstrated that acclimation to heat is impaired by a low-salt diet (6g NaCl/day), and compared to moderate-salt (15g NaCl/day) intake, significantly higher work heart rates, rectal temperatures and incidence of heat injury were noted. In addition, subjects consuming the 6g NaCl/day elicited poorer circulatory responses to postural change and failed to show any improvement in their orthostatic responses during the first few days of heat acclimation (14,15). Taylor and colleagues (14,15) concluded that 15g NaCl/day was required to work under hot conditions where sweat losses would be 5-8 L/day. They further concluded that the 6g NaCl/day or their low-salt diet contributed to a salt deficit and subsequent cardiovascular strain and heat injury. Several others have concurred with the findings that low-salt diets contribute to salt deficits during work in the heat (3,9), salt deficiencies elicit poor circulatory responses to work and postural change (3,16) and the highest incidence of salt depletion and postural hypotension and syncope occur in the first few days of heat acclimation (3,9,16). However, others (3,9,13) have reported optimal salt requirements ranging from 2 to 20g NaCl/day for both acclimatized and unacclimatized men.

Based on these studies, we anticipated a negative sodium balance and a higher incidence of heat injury and postural syncope during the first 3 to 5 days of exercise-heat exposure in subjects consuming the low-salt (4g NaCl/day) diet compared to the moderate-salt (8g NaCl/day) diet. However, after completing 8.5 hrs of intermittent treadmill exercise under hot simulated desert conditions on each of 10 days, all of our subjects completed the orthostatic test without incidence of hypotension or syncopal symptoms. In addition, the pre-exercise heart rate and blood pressure responses to standing were not different between the low- and moderate-salt diets or between heat acclimation days. Our results indicate that prolonged work in the heat can be performed for successive days with consumption of 4g NaCl/day and adequate fluid replacement without increasing the incidence of heat injury or circulatory incompetence.

In spite of the absence of orthostatic hypotension and presyncopal symptoms during the 10 day heat acclimation period, during the post-exercise orthostatic test on day 11 (fourth day of acclimation) significantly smaller increases in diastolic and mean blood pressures at 2 and 4 min standing were observed in the low-salt diet. Concurrent with this poorer orthostatic response was a delay in plasma volume expansion in the low-salt diet during the initial days of heat acclimation (day 11). It is generally agreed that much of the cardiovascular improvements with heat acclimation occur within the first four days of exercise-heat exposure and are due to an increase in plasma volume (2,14). With the increase in plasma volume (days 15 and 17) in the low-salt diet, the post-exercise responses to postural change were

not different between diets. This result is in agreement with several studies that have observed orthostatic hypotension [illegible] dehydration and decrements in [illegible] after rehydration (6).

Pitts and colleagues (10) reported that replacement of sweat losses with water resulted in sustained effective work performance in the heat, and that replacement of salt alone showed no benefit. Furthermore, several studies (10,13,14,15) have noted that the higher the dietary-salt intake, the more fluid consumed. In the earlier studies in which low dietary-salt intake elicited orthostatic hypotension or syncope (3,14,15,16) or increased the risk of heat injury (1,3,9,14,15), fluid replacement of sweat losses was inadequate and significant body weight deficits usually occurred. Salt-depletion heat injury develops over several days and occurs when large volumes of sweat are replaced by adequate fluid intake but not salt (9,13,16). Furthermore, sodium depletion is rarely elicited by dietary restriction of NaCl alone in healthy men in normal climates (16). Thus, it is probable that dehydration significantly contributed to the high incidence of heat illness and syncope observed in the earlier studies (1,3,9,14,15) particularly since circulatory incompetence and orthostatic hypotension are more marked when dehydration accompanies salt deficiencies (16). In the current study, the replacement of all sweat losses incurred during each exercise-heat exposure by an equal volume of pure water on an hourly basis resulted in negligible body weight losses which may have prevented heat injury during exercise and postural hypotension and syncope during the post-exercise orthostatic tests.

Sohar and Adar (13) conjectured that the majority of salt-depletion heat injuries reported in the literature most probably were not actual salt deficiencies but rather represented the inability of unacclimatized individuals to conserve salt and replace water losses during the first few days of heat acclimation. It is generally accepted that salt losses in the urine and sweat are dramatically reduced by heat acclimation (1,2) and maximal conservation occurs by 5 and 10 days, respectively. In the current study, sodium conservation was evident in both the 4 and 8g NaCl diets; urine and sweat sodium losses were significantly reduced during the first four days of acclimation in the moderate-salt diet and during the entire acclimation period in the low-salt diet (Moore et al., unpublished data). Francesconi et al. (6) demonstrated that endocrinological adaptations, especially in the low-salt diet reflected renal and sweat gland NaCl conservation contributing to a positive sodium balance throughout most of the acclimation period.

Acclimation to heat has been reported to both enhance (5,11,12) or have no effect (4,7) on orthostatic tolerance in either warm or hot climates. Our results suggest that heat acclimation had no effect on blood pressure and heart rate responses while either supine or standing, irrespective of dietary NaCl intake. Although one of the important effects of acclimation on orthostatic responses may be a reduced incidence in syncope following work in the heat (5,7,11,12,14), the absence of syncopal symptoms in the current study did not enable us to address this hypothesis.

Our results indicate that while consuming 4g NaCl/day with adequate fluid replacement, prolonged work in the heat can be performed on successive days without orthostatic hypotension or syncope. We further conclude that the blood pressure and heart rate responses to postural changes showed no improvement with heat acclimation irrespective of dietary-salt intake. Future research should evaluate the effects of combined NaCl restriction and ad libitum water consumption on orthostatic responses after prolonged work in the heat.

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